THE AMERICAN JOURNAL
OF
OPHTHALMOLOGY

VOL. XXVII. NOVEMBER, 1910. No. 11.

ORIGINAL ARTICLES.

COQUILLE PROTECTIVE SPECTACLES:

RELATION OF THE THICKNESS OF A COQUILLE OF ZERO POWER TO THE PRINCIPAL FOCAL LENGTHS AND TO THE POWERS OF ITS TWO SURFACES.*

BY JOHN GREEN, M.D.,
ST. LOUIS, MO.

[Adopting the current *metric* notation, in terms of *dioptries,* the function μ (the *index* of refraction) drops out of the several equations, and the demonstration is reduced to a discussion of three geometrically related linear values.]

The point F (Figure 1), plotted as the limiting position of q for decreasing magnitudes of the angle

$$Q\,O_1N_1=Q_n\,B_1N_1=\phi,$$

represents the common principal focus of the two spherical surfaces of which A_1F, A_2F represent the principal focal lengths.

By construction,

$$A_1A_2=A_1F-A_2F;$$

or, designating these several linear values by d, f_1, f_2,

$$d=f_1-f_2;$$

which defines the thickness of a coquille of zero power, at its centre, as the arithmetical difference of the principal focal lengths of its two surfaces.

*Communicated to the St. Louis Ophthalmological Society, as an addition to a demonstration presented January 25, 1909, and contributed to "The American Journal of Ophthalmology," November, 1909.

Again, writing $\frac{1}{D_1}, \frac{1}{D_2}$ for f_1, f_2,

$$d = \frac{1}{D_1} - \frac{1}{D_2} = \frac{D_2 - D_1}{D_1 D_2} \quad \ldots\ldots\ldots\ldots (A)$$

As D_1, D_2 are reciprocal to f_1, f_2, they represent, *in dioptries*, the respective powers of the two surfaces of which f_1, f_2, *expressed in metric notation*, are the principal focal lengths. In the form,

$$d = \frac{D_2 - D_1}{D_1 D_2},$$

the equation defines the thickness of a coquille of zero power, at its centre, as the quotient of the arithmetical difference of the powers of its two surfaces divided by their arithmetical product.

From the form of the second member of the equation it is obvious that the numerator and the denominator of the fractional expression are differently affected by variations in the values D_1, D_2. Assuming, for instance, a very small real value for D_1, and supposing D_1 and D_2 progressively increased by identical additions to each, the value of the numerator remains unchanged while with progressively increasing values of the denominator the value of d, as expressed by the fraction as a whole, progressively decreases. In other words, for any constant difference of the powers of its two surfaces a coquille of zero power may be imagined of any finite thickness and, conversely, for any assumed thickness of the coquille the powers of its two surfaces may have an indefinite number of conjugate values.

Inasmuch as increasing values of D_1, D_2 connote decreasing values of f_1, f_2, which are equimultiples of r_1, r_2, it follows that:

(*a*) For any assumed difference of the powers of its two surfaces the thickness of a coquille of zero power decreases for every increase in its curvature.

(*b*) For any assumed thickness of a coquille of zero power the difference of the powers of its two surfaces increases for every increase in its curvature.

EXAMPLES:

Table I gives the thickness of a protective coquille as computed for differences in power at its two surfaces ranging from 0.25 *dioptrie* to 2. *dioptries* and for lower, medium, and higher curvatures. Of the twenty-four cases shown in the table, thirteen, marked (*), present combinations satisfying possible practical requirements. The number of such examples may be increased *ad libitum* by varying the numerical values assumed for D_2 and D_1.

TABLE I.

$$d = \frac{D_2 - D_1}{D_1 D_2} \text{ metre.}$$

$D_2 - D_1 =$ dioptrie	$D_2 =$ dioptries	$D_1 =$ dioptries	$d =$ metre
0	n	n	$0/n^2 = 0$
0.25	5.	4.75	0.25/23.75 = 0.010 526*
	10.	9.75	0.25/97.5 = 0.002 564*
	15.	14.75	0.25/221.25 = 0.001 129*
0.5	5.	4.5	0.5/22.5 = 0.022 222
	10.	9.5	0.5/95. = 0.005 263*
	15.	14.5	0.5/217.5 = 0.002 298*
0.75	5.	4.25	0.75/21.25 = 0.035 294
	10.	9.25	0.75/92.5 = 0.008 108*
	15.	14.25	0.75/213.75 = 0.003 508*
1.	5.	4.	1./20. = 0.050 000
	10.	9.	1./90. = 0.011 111*
	15.	14.	1./210. = 0.004 761*
1.25	5.	3.75	1.25/18.75 = 0.066 666
	10.	8.75	1.25/87.5 = 0.014 285
	15.	13.75	1.25/206.25 = 0.006 060*
1.5	5.	3.5	1.5/17.5 = 0.085 714
	10.	8.5	1.5/85. = 0.017 647
	15.	13.5	1.5/202.5 = 0.007 407*
1.75	5.	3.25	1.75/16.25 = 0.107 692
	10.	8.25	1.75/82.5 = 0.021 212
	15.	13.25	1.75/198.75 = 0.008 805*
2.	5.	3.	2./15. = 0.133 333
	10.	8.	2./80. = 0.025 000
	15.	13.	2./195. = 0.010 256*
n	n	0	$n/0 = \infty$

A CASE OF BRAIN ABSCESS WITH RARE OCULAR SYMPTOMS.

By Dr. C. Barck,
St. Louis, Mo.

The patient, Mrs. V. C., was seen by me for the first time on the 10th of January, 1910. She had been married for two years, but had no children. One brother and two sisters are living and healthy.

Previous history: Patient had a congenital, slight facial paresis on the right side. It had been noticed by her parents, soon after she was born. Since childhood, she was suffering from a chronic otorrhœa of the left ear. This was attended to at times, but in the main neglected. There was no pain in the ear, until the beginning of 1909. The pain increased by and by, and became very severe in March and April of the same year. The discharge was copious and in May the mastoid region commenced to swell. On the 26th of May a mastoid operation was performed at her home in the country. But after the first week, the severe pain reappeared. It was located at that time mainly in the back of the head and neck. On account of this persistent pain, a second operation was performed two weeks later. The physician who was present during the procedure, stated that a kind of radical operation had been done and that the ossicles had been removed. But the pain in the head continued. It was felt partly in the forehead and partly in the occipital region. Soon after the second operation, vomiting set in and during a period of three to four weeks the patient vomited after every meal. Then it gradually subsided.

One week after the second operation, the patient noticed that her vision was impaired; but no examination was made at that time. She stated that the condition was a changeable one; that at times she could see quite well, then again everything would appear dim. Since September, 1909, a decided diminution in the visual acuity took place, and six weeks prior to her consultation with me she noticed—and her family physician found on rough examination—that she had hemianopia. She was not able to see within the right half of the field of vision. Patient was still able to read on the 1st of November, 1909, but not since.

Examination: No discharge from ear. Large, dry cavity. Drum-membrane and ossicles wanting; but the posterior wall

in its entirety present, proving that no radical operation could have been performed. In the mastoid region a large, irregular scar, somewhat depressed. No fistula; no redness around the scar. No pain on moderate pressure. Generally speaking, it was the condition we find after the healing of a mastoid operation. But there was one spot, about one inch above and one inch behind the scar, which was very painful to pressure, as well as to percussion with the style (a method recommended by me some years ago). The painful area was an irregular circle, with a diameter of about two-thirds of an inch. On repeated examinations by different observers, this painful site was invariably discovered. It corresponded to about the posterior portion of the temporal, or to the foremost portion of the occipital lobe.

Spontaneous pain was not experienced within this area; but was located farther back in the occiput. There was no pronounced stiffness in the muscles of the neck, but movements of the head, especially forward or bending movements, were very painful. There was no dizziness and never had been. Examination of the labyrinthine functions proved them intact.

The vision of the right eye was 1/60; that of the left 1/36; could read none of Snellen's test types. There existed complete right homonymous hemianopia. The division line was vertical, passing through the point of fixation. There was no palsy or paresis of the external ocular muscles. The ophthalmoscope revealed a choked disc of considerable degree in both eyes. The difference between the height of the swelling and the surrounding retina was measured as 3 diopters, corresponding to an elevation of 1 mm. In the left eye there were two large hæmorrhages close to the optic papilla, one of them having a diameter as large as that of the disc. No hæmorrhage in the right eye.

The paresis of the right facial nerve, mentioned in the anamnesis, was of a moderate degree. It was confined to the lower branch of the nerve, controlling the muscles around the mouth, and became most apparent when the patient laughed. The upper branch, controlling the orbicularis muscle, was intact. Relatives made the statement that this paresis had become more manifest the last month (which statement was at the time accepted "cum grano salis").

Speech and memory were not affected. The patient spoke fluently, rather rapidly. She answered all questions in a precise manner. Her physician stated, that in June her memory had been somewhat defective; that she could not remember

names correctly; but the report was too vague to be of any value. The mental state of the patient, however, was an abnormal one. It was one of labile psychical equilibrium. As a rule she was very much depressed and wept frequently. Moreover, she was very excitable, so that the examination had to be interrupted at times.

During the next few days of observation the pulse-rate varied between 80 and 100, whilst the temperature was always found to be normal.

I made the diagnosis of an otitic brain abscess in the left temporal lobe, reaching far back, so as to encroach upon the optic radiation (Gratiolet's visual fibres) in the posterior part of the capsula interna. The symptoms seemed to me sufficiently clear, to assume the presence of a cerebral abscess and the only question to my mind was that of the different diagnosis between the location in the temporal lobe or in the cerebullum. Hemianopia could be the consequence of a lesion in either; in the first instance by affecting the optic radiation or the optic tract; in the second by injuring the cortical center in the occipital lobe, either by transmitted pressure or by direct destruction after perforation of the tentorium. Such a case is on record. The fact, that the dividing line between the preserved and the destroyed halves of the fields of vision passed through the point of fixation, may have been accepted in favor of the former view. For, according to some authorities, the dividing line, in lesions of the optic tract, is a vertical one and the central vision in consequence materially impaired. In lesions beyond the primary centers (geniculate bodies) the dividing line passes in a curve around the center of the field, so that the macula region is spared and the central vision very good, often normal. This theory, however, is not yet generally accepted; and furthermore the presence of the enormous choked discs complicated and obscured the clinical picture to such an extent, that I did not feel justified, to draw any conclusions from these findings of the perimeter.

My diagnosis of a localization in the temporal lobe rested mainly on the following points: The circumscribed area, which was so decidedly painful to percussion; the want of cerebellar symptoms, as ataxia, dizziness, etc.; and the absence of suppuration of the labyrinth. At first, on noticing the facial paresis of the right side, I thought that this symptom might also be used for topic diagnosis, speaking again in favor of localization in the temporal lobe; but after learning that it was congenital, it had to be left out of consideration.

During the following days a number of consultations were held, with an oculist, Dr. J., and a neurologist, Dr. G. The opinion of the former coincided with my view; but the latter, whilst not denying the possibility of a suppurative process, maintained the probability of a chronic encephalitis, basing his opinion mainly on the congenital paresis of the facial nerve. Yet, under the existing circumstances, he considered an operation justified.

Operation: Performed on the 14th of January. The section was made through the old scar and continued upward and backward. In the exposed mastoid there were still some small carious foci; but the bone was generally firm. The posterior wall, towards the cerebellum, consisted of perfectly healthy, dense bone. The supramastoid crest, the tegmen antri and the postero-inferior portion of the squama of the temporal bone were then removed, to expose the middle cranial fossa. The bone of these regions was enormously sclerosed and hard like ivory, requiring tedious work with the bone-forceps. An area of the dura, about one inch in diameter, was laid bare. The upper and posterior portion of this exposed area showed a spot of marked discoloration, which corresponded quite closely to the painful site found on percussion. After incision of the dura, the brain substance bulged out under considerable pressure. At the spot mentioned, the dura was adherent to the cerebrum. The temporal lobe was then explored, partly with a long narrow knife and partly with a trocar. I introduced the instrument about twelve times in different directions, going in as far as 6 cm. Special care was taken, to go backward and upward as far as permissible, and I came in contact with the tentorum several times. But no pus was encountered. A drainage tube, 5 cm. long and 1 cm. in diameter, was inserted and the usual dressings applied.

The subsequent course, for the first four weeks after the operation, was a very favorable one. The immediate effect of the release of the intracranial pressure was remakable. The pain in the head disappeared completely and the entire mentality of the patient became changed. The psychic depression had given way to a happy, at times even hilarious, disposition. She was convinced that she would recover. There was no rise of temperature; sleep and appetite were excellent. The fundi were examined every few days; three days after the surgical interference a considerable decrease in the size of the choked discs was noticed and they gradually became smaller during the following weeks. Within this period the vision of the left eye improved to a certain extent, so that the patient could read Snellen XII and later

on XI with difficulty. The sight of the right eye did not improve materially. The hemianopia remained always the same.

On the second dressing it seemed that there was some pus in the tube; but none could be discovered subsequently. As was to be expected, a cerebral prolapse of medium size formed; this had to be clipped on two different dates, on the 24th of January and on the 9th of February.

The patient had been out of bed for some time, when a gradual change in the clinical picture took place. On the 7th of February the temperature rose to 100 and kept on rising during the following days, reaching 103-104. The headaches reappeared, becoming so severe, as to necessitate the administration of anodynes. On the morning of the 14th there was loss of speech (amnestic aphasia). Then coma set in, which increased rapidly until the exitus letalis on the 18th of February.

Post Mortem: The brain was removed and upon the advice of Dr. G. put into formol solution, to be cut into sections afterwards. There was a moderate degree of lepto-meningitis, more pronounced over the left side. Furthermore, there was a fresh hæmorrhage in and around the Sylvian fissure. Whether or not an abscess of the brain was present, could not be determined at the time of removal. When the specimen was sectioned, ten days later, by Dr. G., Dr. Th., pathologist, and myself, a cerebral abscess was found. It was relatively small, about three by four cm. in diameter, and surrounded by a thin capsule, which was partly broken. The pus was almost odorless; whether this was due to the formol, or whether it had been inoffensive primarily, I am unable to state. The abscess cavity was located in the extreme postero-superior portion of the temporal lobe. After placing the sections of the brain together, we measured the distance of the abscess from the point of incision, and found it to be 6½ cm. I am, of course, aware of the fact, that after these manipulations with the specimen, the findings are only approximately correct; yet we were very careful to restore the normal condition as far as possible.

Remarks: The clinical picture was somewhat obscured by the fact, that the crossed facial paresis was a congenital one. Had it been acquired, it would have been a valuable symptom, proving a lesion of the capsula interna. It furthermore weakened to a certain degree the conclusions, which could be drawn from the hemianopia. The subsequent improvement of the paresis placed the mentioned statement of the relative in a different light; and the inference from this improvement in view of the autopsy is,

that the previously existing paresis was increased by the pressure of the brain abscess and improved after this was relieved. But apart from the facial paresis, the hemianopic symptoms were so definite, that I assumed, on this basis, an encroachment on the visual radiation in the capsula interna. In spite of the vertical dividing line of the field of vision, I did not consider an affection of the optic tract probable. I furthermore concluded, that the abscess must be a very large one, in order to reach so far back. Moreover, this conclusion seemed to be corroborated by the enormous degree of the choked discs; such as I have never seen before in brain abscess.

There are about twelve cases on record in literature, where otitic abscesses of the temporal lobe caused hemianopia. But in none of them is it stated, whether the dividing line of the field of vision passed through the very center, or whether the macula was spared.

The retrogression of the choked discs after the release of the intracranial pressure was a very rapid one; and if conclusions may be drawn from a single experience this would speak in favor of the "pressure theory," against the "toxin theory" of choked disc. Since the one factor, the tension, was removed by the operation, whilst the other, the abscess, remained undisturbed, the lesson learned from the case is equivalent to that of a physiologic experiment.

Otitic cerebral abscesses are, as a rule, found close to the primary focus in the bone. In this case the distance was unusually far, and even the path of infection could not be detected at autopsy. There is but a single case on record of an abscess at the distance of 7 cm. from the surface, which was found and opened surgically; the termination was fatal, because of the impossibility to establish proper drainage. For this very reason I believe, that also in my case the outcome would have been the same had the abscess been opened. The advice given by Koerner and others is: not to enter with instruments farther than 4 cm., in order not to injure the lateral ventricle. As previously stated, I introduced the knife considerably farther. During the after-treatment I felt inclined to probe once more, but as I had about abandoned the diagnosis of a suppurative process, I considered it contraindicated. The aphasia, which occured shortly before death, is of course explained by the recent hæmorrhage in the region of the Sylvian fissure, affecting the center of speech.

A CASE OF LYMPHANGIO-ENDOTHELIOMA OF THE LOWER EYELID.

By Adolf Alt, M.D.,
St. Louis, Mo.

October 3d, 1910, Mrs. X., almost 60 years old, came to my clinic at the St. Louis Mullanphy Hospital on account of an unsightly tumor of the right lower lid. The patient stated that six years previously a "cyst" had been removed from the same place, having been in existence for some years previously, at some physician's office.

The outer half of the lower right lid was now the seat of a tumor about the size of a small walnut. It was covered with

Figure 1.

very thin, atrophic skin, which to some extent could be moved over it. The shape of the tumor was roundish, but several harder projections and nodules on the visible surface gave it an appearance like a "bulbus quadratus." The tumor was slightly movable on the underlying tissue and on the whole made the impression of an uncommonly large cyst, perhaps a sebaceous cyst. This diagnosis seemed the more probable since the palpating finger got the impression of semi-solid contents, with a doubt-

ful fluctuation. The patient consented the more readily to the removal of the tumor since its weight dragged the lid down and caused lacrimation, and since it was unsightly.

I made the enucleation of the cyst under ether narcosis and succeeded in removing it in toto without rupture of the sac, an accident which I was almost prepared to see happen, since it was so thin at the outer surface. The wound was closed with five stitches and healed per primam.

The specimen was put into a 5 per cent. formol solution. When I examined it the following day, manipulating it with two forceps, I ruptured the sac and in the next few days the shrink-

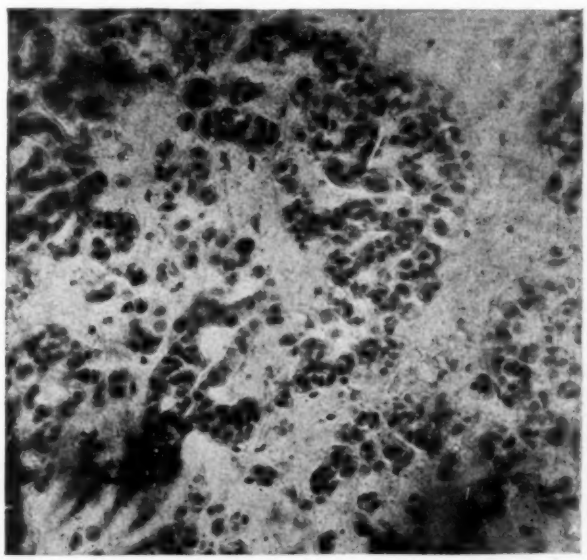

FIGURE 2.

ing of sac extruded most of its contents, which lay beside the sac at the bottom of the bottle as if there were 2 tumors almost of equal size. The contents formed a semi-solid grayish yellow substance. During the hardening process this substance shrank gradually to less than half of its original size by the withdrawal from the tissue of its fluid parts, which it must have contained in great quantity.

The microscopical sections stained with hæmatoxylin-eosin gave a very peculiar picture, such as I could not remember ever having seen before. (Fig. 1.) Distributed irregularly in a transparent and almost unstained, evidently very œdematous con-

nective tissue were rows, patches and sometimes rings of epithelium-like cells, which, however, appeared very strange. Single and double rows and sometimes circles of roundish and oval nuclei seemed to be contained in continuous columns and bodies of protoplasm, so that no division into single cells could be made out. This arrangement of the cells resembled nothing so much as the syncytial cells of the chorion papillæ. (See Figures 2 and 3.) The round bodies with many nuclei did not give the impression of giant cells, but were apparently only smaller fragments of the same cell bodies which made up the longer columns.

The few bloodvessels found in the tumor were almost per-

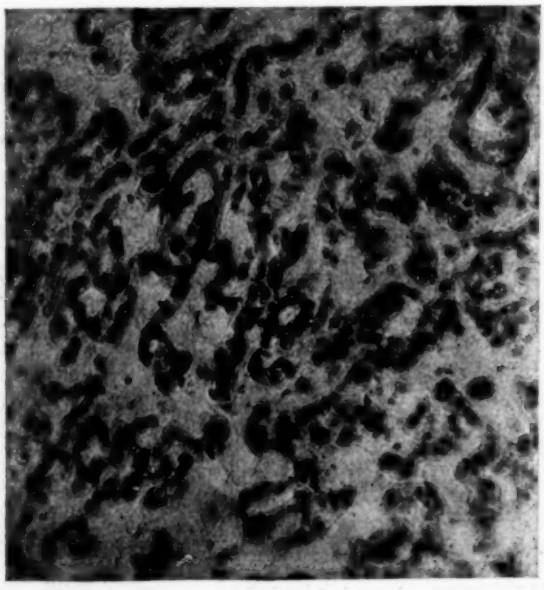

FIGURE 3.

fectly ensheathed by these peculiar tumor cells in single or double rows.

The stroma in which these cell columns were embedded consisted in the main of a still quite œdematous connective tissue, although it had shrunken so much during the hardening process. Staining with phosphotungstic hæmatoxylin showed a beautiful network of connective tissue fibres.

In some parts there were numerous oblong cells with several offshoots at the poles and sideways which could be traced for some distance; here the stroma had taken on a little more of the

eosin stain and the connective tissue was evidently taking on a myxomatous character. (See Fig. 4.)

The harder projections and nodules in the outer part of the cyst sac were made up of cartilage tissue (see Fig. 5), surrounded by the peculiar cell columns.

Instead of being a sebaceous cyst the tumor, therefore, presented much more the aspect of a mixed tumor. Yet, whence had it arisen and what was its true character?

After hunting in vain through the literature at my disposal, I was finally made acquainted with a paragraph in E. Lexer's General Surgery (translated by A. D. Bevans, 1908), page 904,

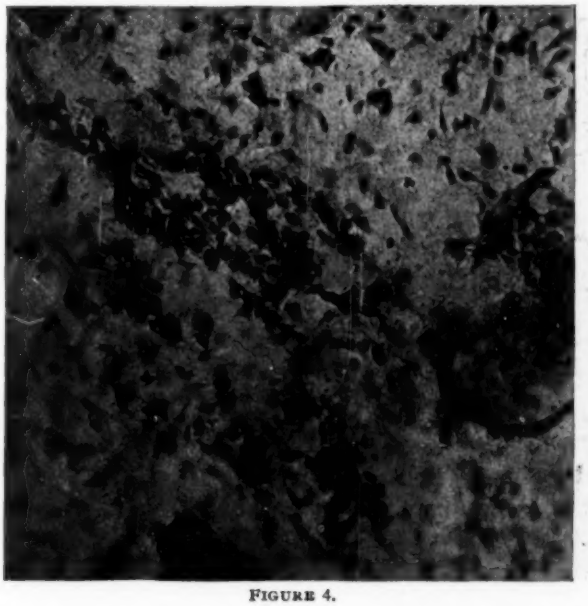

FIGURE 4.

which in all its details fitted the tumor under consideration admirably. The illustration of Lexer (Fig. 437) might even have been drawn from my specimens.

Here is what Lexer says:

"Lymphangio-endotheliomas develop from the endothelial lining of the tissue spaces and lymphatic vessels. These tumors are composed of columns of cells which correspond to the course of the lymphatics. The cells which have a tubular or solid arrangement are flat, cubical or columnar. The three varieties are found in different parts of the tumor or combined in the same

field. The cell columns are enlarged at the nodal points and cells composing them may be concentrically arranged. According to Borst the cell columns, which differ in size, have a delicate linear arrangement and are lined by two layers or a single layer of cells resembling very closely the histological picture of proliferating lymphatic vessels. These tubules may dilate to form cysts. If papillary growths develop the tumor may be mistaken for a cavernous or cystic lymphangioma or for a papillary cystadenoma (e.g., of sweat glands).

"*Stroma.*—The character of the connective tissue stroma varies. It may be very cellular, mucoid or fibrous. Probably a meta-

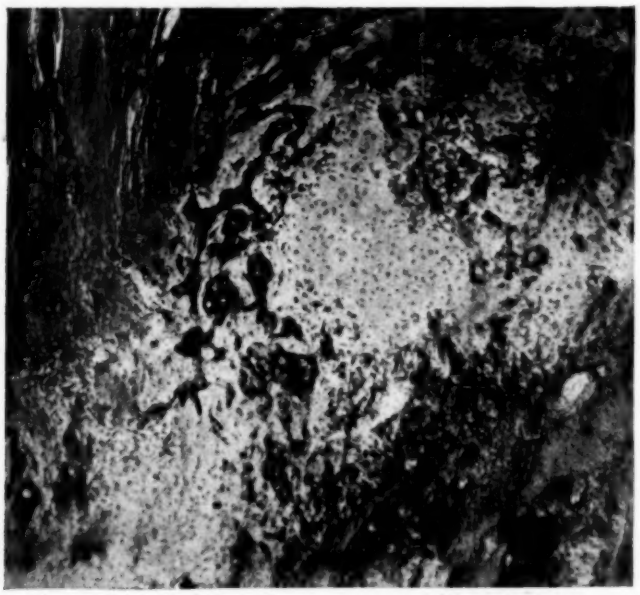

FIGURE 5.

plasia of the stroma into cartilage, occasionally even into bone, occurs, the presence of cartilage and bone in mixed tumors of the parotid and related forms of endotheliomas being explained in this way.

"*Most common sites for development.*—Lymphangio-endotheliomas of the skin and subcutaneous tissues, especially of the face, appear as circumscribed, encapsulated nodules or non-encapsulated growths which are sometimes regarded as carcinomas, sometimes, especially when ulcerated, as carcinomas of the skin. They grow slowly, but rarely forming metastases, and

therefore are to be regarded as relatively benign tumors. According to Tanaka, the lymph nodes when involved are of a very soft, fluctuating consistency and not adherent to the surrounding tissues."

The tumor above described grew in the region of predilection of these tumors, the face. It had taken six years to attain the size of which I found it, or if the previously removed "cyst" was in reality of the same nature, it had, with an interruption, grown even for a longer time without, so far as I am able to find, having caused any metastasis. No swollen lymph glands could be detected near it. The tumor was encapsulated.

The histological appearances correspond in every way with the description given by Lexer. There seems, therefore, to be no doubt as to the character of our tumor. It is evidently a rare specimen of a lymphangio-endothelioma of the eyelid.

DOUBLE CENTRAL BLINDNESS FOLLOWING INJURY TO THE HEAD BY A FALL.

L. Buchanan (*Glasgow Med. Jr.*, October, 1910) reports a case of a man who fell on the street striking the back of his head on a stone which resulted in the loss of central vision in both eyes, the peripheral vision being but slightly, if at all, affected. The vision of the right eye was but 3/60 and of the left 1/60. Perimetric examination showed but little contraction of the visual field but a distinct, though small sized, scotoma in each eye.

BLINDNESS DUE TO THE ADMINISTRATION OF SYNTHETIC ARSENICAL COMPOUNDS.*

By F. L. Henderson, M.D.,
ST. LOUIS, MO.

I find cases of toxic amblyopia from the administration of synthetic arsenical preparations have been reported since 1905. Owing to the rapidly increasing number of agents held responsible for amblyopia, the reports of atoxyl blindness made no special impression on my mind. Assuming that you may have given this subject no more serious consideration than I did, I wish to emphasize its importance. This question has already been brought to our attention by Dr. A. E. Taussig, in the August number of the *Interstate Medical Journal,* but I have been made to feel it will bear repetition by meeting with the unfortunate case which I have to report.

March 16th, 1910, a man walked into my office with the unmistakable gait of a tabetic. He complained that his vision was failing. He was 50 years old, and 16 years before a condition which was diagnosed and treated as rheumatism began. Five years ago the diagnosis of tabes was first made. Reading glasses were prescribed at 46, but he felt no need for distance lenses until 48. He was using plus 1. D.S. in each eye for distance and plus 3.25 D.S. for near. He said his vision had failed perceptibly in the past two weeks. He was exceedingly nervous. His expression was anxious and care-worn. His general appearance was frail and anæmic. The visual test was as follows: Right eye with plus 1.75 D. S., combined with minus .25 axis 30 equaled vision of 6/6 minus. Left eye, with plus 1.25 D.S., combined with minus .25 axis 90 vision equaled 6/6. With both eyes corrected, vision was 6/5. With plus 2.25 D.S. added near vision was normal. The muscle test with Maddox rod showed a very slight exophoria. The ophthalmoscope revealed a large deep excavation in the right disc, glaucomatous in appearance; the left disc was pale but not excavated. Tension of each eye was normal. On March 18th, two days later, he accepted the same correction for the right eye and got the same vision. The left eye showed some slight change in the correction, but the best vision obtainable was 6/9 minus. The fields of

*Read at the meeting of the St. Louis Ophthalmological Society, Nov. 14th, 1910.

vision were taken and both found concentrically contracted to about 15 degrees. He could not stand a perimetric examination and the fields were obtained roughly with a white target on a black rod. Central color perception was normal. Both pupils responded feebly to light and convergence. The patient insisted that his vision was getting rapidly worse. In view of the fact that his central vision was still nearly normal and that two days previously it had been 6/5, I concluded that the change which he noticed was due to rapid contraction of the fields. About this time a consultation with his neurologist revealed the fact that the patient had only lately returned from New York, where he had been treated by hypodermatic injections of soamin (para-amino-phenylarsenate), a preparation containing 22.8 per cent. of arsenic. His physician here had continued the injections only after explaining its dangers and advising against its use. This treatment had been discontinued a short time before the patient reported to me. The dose given by the New York physician was one tablet every other day until 5 were given. The St. Louis physician gave him but one injection of ¼ of a tablet. This was all I could learn of the amount, which is exceedingly unsatisfactory in view of the fact that soamin is put up in 1 gr. tablets and 5 gr. tablets. I prescribed pills hydrarg. protoiodide, grain 1/4 each, three times a day and administered strychnia sulphate hypodermatically daily, beginning with 1/20 of a grain. On March 20th vision of the right was 6/7½ and left 6/9 minus. On the 21st the patient was in such a state of nervous exhaustion that he remained in bed. When seen at his home vision was alarmingly worse, amounting to an indefinite determination of objects. That night he took a train to Hot Springs to consult an oculist in whom he reposed great confidence. He returned in four days totally blind. I renewed the treatment by protoiodide internally, and ran the daily injection of strychnia up to 1/7th of a grain. Central vision improved for a while but varied greatly from day to day; then slowly faded into a state of hopeless blindness. My services were no longer needed but were continued at the request of the patient's wife and his attending neurologist, who thought it best, owing to the patient's depressed condition, to postpone a hopeless visual prognosis as long as possible. At this time he ran afoul of an advertising genius in Chicago who had cured (?) a large number of prominent people in the last states of locomotor ataxia. One of the grateful patients even came from Chicago to tell him of the wonders

being wrought by this health-giving magician. When it became evident the patient was going to Chicago, I had a talk with his wife in which I described to her the methods by which they would be separated from their money before the great tabetic specialist would undertake the case. My purpose in fore-arming her was to reduce the loss from the impending robbery to the lowest denomination in my power. I wonder now what it would have been had they stumbled into the ambush without warning. The patient returned in two weeks, on a stretcher, much reduced in strength and spirits, in no way relieved except of the paltry sum of $4,000.00 in cash. He brought back nothing except the definite assurance that great improvement would be observed in exactly ten days. I refused to see the patient again as he was still in communication with the Chicago tabetic specialist, and as he was past any service from me. The predicted relief came, but not exactly on schedule time, as he did not die till October 10th.

Was blindness due to simple tabetic atrophy or was it tabetic atrophy disastrously influenced by the treatment? A reference to the literature of the arsenical treatment of syphilis of the nervous system inclines one to the latter opinion. A number of arsenical preparations have been introduced in the last few years. Among them are atoxyl, arsacetin, soamin and orsudan. If interested in the incomprehensible, compound names by which these substances are known to chemistry, such information can be gotten from the trade catalogues of the manufacturers.

In 1905, Bornemann reported a case of blindness resulting from the ingestion of 27 grams of atoxyl. In 1906, Krudner reported a case of blindness in a patient who took 50 grains of atoxyl in the course of 7 months. Both nerves became atrophic, one eye became blind and vision in the other was reduced to 1/15. The fields were greatly contracted.

In 1907, Fehr reported two cases which presented marked contraction of the nasal fields, atrophic color of the nerve heads and contraction of the arteries. One patient received 25 grams of atoxyl during 16 days and the other 10 grams while under treatment for pemphigus. Hollopeau reported one case of alcoholic neuritis in which blindness followed an intra-muscular injection of atoxyl. Vision began to fail several days after its use and in 14 days sight was gone. In 1908, Nonne reported one case of blindness from atoxyl. Herford reported two cases of blindness and one of retinal hæmorrhage from atoxyl. In 1909, Coppez re-

ported a case of total blindness from 5 doses of atoxyl, 5 centigrammes each. Rosenfeldt reports one case of atrophy from atoxyl. Paderstein also reports a case. Kalaschnokow reports a case of syphilis treated by ten injections of a 10 per cent. solution of atoxyl. The result was optic nerve atrophy beginning with concentric narrowing of the visual fields. After the deleterious influence of atoxyl had been proven arsacetin was introduced, and Erhlich, Neisser, Heymann, and G. Klemperer emphasized the fact that affections of the optic nerve were never observed after its use. Since then Judin has reported a case of total blindness from optic nerve atrophy as a result of 4 injections of arsacetin. Ruete produced atrophy of the optic nerve by six injections of arsacetin given for psoriasis. Weinstein reports optic atrophy after two injections of arsacetin. Iverson had one case of blindness following the use of arsacetin for relapsing fever. The patient had but two doses seven days apart. Hammes reports a case of anæmia in a patient of 66 years treated by 8 subcutaneous injections of 0.1 gram arsacetin. In three weeks patient was almost blind from retro-bulbar neuritis.

Later soamin was said to be free from the evils attendant upon the use of its predecessors; but at the June, 1910, meeting of the Ophthalmological Society of the United Kingdom, Mr. E. Clark reported the case of a man aged 46, treated for syphilis. After ten injections of soamin, 5 grains each, vision began to fail. Five months later he was given 5 more injections and after the third vision began to fail again. Vision of one eye is reduced to shadows and the other to 6/12. He reports another case treated with orsudan in which vision was reduced to counting fingers after 9 injections. Mr. Clark also referred to cases described by Ward, Bagshaw, Someren and Luedie. I am not prepared to say what preparation was used in these cases as I have not seen a printed report of them.

The arsenical preparations have been found of great value in combating trypanisomiasis (sleeping sickness), but the result to vision has in many instances been disastrous. Koch in his observations on sleeping sickness reports 22 cases of blindness among those treated with atoxyl. Ayers-Kopke saw 6 cases of double optic atrophy out of 29 patients treated for sleeping sickness with atoxyl. Beck gives an account of 900 cases of sleeping sickness treated by sub-cutaneous injection of atoxyl; 23 were made totally blind and 7 suffered more or less loss of vision. Eckard reports three cases of blindness out of 134 treated for sleeping sickness with arsacetin.

The following experiments and conclusions in reference to the action of these drugs upon the visual apparatus are taken from the Ophthalmic Year Books of 1908, '09 and '10. Fehr concludes that repeated as well as single large doses of atoxyl can produce optic nerve atrophy, which may pass on to complete blindness, or, if the toxic agent is withdrawn in time, result in recovery. The beginning of the visual disturbance can come on gradually, or appear suddenly and general intoxication symptoms need not necessarily accompany or precede the ocular disorder. In the cases thus far observed, in the beginning there is concentric, especially nasal, contraction of the visual field, without central scotoma, and quite early pallor of the entire nerve-head, with narrowing of the retinal arteries. The condition seems to depend upon a peripheral optic nerve process, with alteration in the vessels. Igersheimer has investigated the effects of atoxyl on animals. Injections into the anterior chamber of rabbits produced no permanent pathological conditions. On the other hand injection of 1 mg. and more of atoxyl into the vitreous caused beginning degeneration of the ganglion cells and optic nerve, a proof that by direct contact even in small doses it has a necrotic effect on the nervous elements of the retina. While dogs showed very few general symptoms, after subcutaneous injection, cats developed slowness of movements, ataxia, spasms and spastic paraparesis. The eyes, outside of conjunctivitis and some toxic pupillary phenomena, showed nothing abnormal. Anatomical examination discovered positive though not marked changes in the retinal ganglion cells (chromatolysis, nuclear distortion and death of the cell); and a remarkable change in the optic nerve, characterized by an intense blackening of the nerve sheaths, which has been described by Schreiber as the "Marchi-reaction". The central nervous apparatus also showed cell changes in the brain and cord, and moderate Marchi-degeneration in the peripheral nerves. He could not determine whether the effect in the eye was a primary one, upon the retinal cells, or a secondary degeneration of the retina and optic nerve. In a second paper the same author states evidence educed from animal experimentation goes to show that atoxyl attacks nervous tissue as such; that the primary attack may occur at various points; sometimes it affects the retina, other times the optic ganglia and still other times the optic nerve. Several points may be attacked simultaneously. The amblyopia may have reached an extreme degree before marked ophthalmoscopic changes present themselves. On the other hand the papilla may

be quite white and good vision be retained. The toxic action results from the chemical combination and not from either of the constituents. In very chronic poisoning, however, the arsenic may play a role as in time it becomes split off from the atoxyl molecule in the body. This explains the predominance of the arsenical toxic symptoms in the case of Fehr, and also explains the Marchi reaction in the optic nerve in the long chronic cases. Igersheimer gives the effects of the drug on the general system in acute and chronic toxæmia. Hæmorrhages in the kidneys almost invariably occurred, which are attributed to a primary effect upon the circulatory mechanism of the organ. In sub-acute and chronic poisoning a train of nervous symptoms resulted, slowness of movement, ataxy, clonic twitchings and more or less severe spasms and spastic paralysis. These symptoms result from severe degeneration in the processes of the cells of the brain and spinal cord. The experiments gave no support to the view that the action of atoxyl is due to the anilin phenyl compound. Birch-Hirschfeld and Koster describe the lesions, produced by the daily administration of small doses of atoxyl to dogs and rabbits, to be destructive processes in the optic nerve, retina and central nervous system. Their findings confirm the clinical picture observed in two cases of atoxyl blindness with atrophy of the optic nerve, but without impairment of the pupil reactions. Nonne has made an anatomical examination of the eyes in a case of blindness due to atoxyl. The patient died of carcinoma cachexia. He found a retrobulbar neuritis, of a sub-acute degenerative character, without true inflammatory changes, so that the atoxyl in this case had a direct toxic action upon the optic nerves. The drug had been given continuously in small doses, but produced blindness probably as a result of the general weakness due to the carcinomatosis. Morax has collected the reported cases of ocular symptoms produced by atoxyl. In these cases the drug was usually employed for the treatment of a chronic skin affection or for trypanosomiasis. The ocular manifestation has always assumed the character of an atrophy of the optic nerve, with or without atrophic pallor of the disk. The visual symptoms set in rapidly and attain their height in a few days. There is always a contraction of the visual field, with reduction in the acuity of vision or total suppression of all perception. When the lesions are once produced, they remain stationary, but partial recovery may occasionally occur. The toxic dose is variable, and this uncertainty is the worst feature in the therapeutic use of atoxyl.

Key experimented with atoxyl on dogs. He divides the effects

obtained into two classes: (1.) Acute intoxication in which death occurred in less than ten days. The histological findings were atrophy of the nerve fibres, and possible septal thickening; atrophy of the nerve fibre layer, and diminution of the number of gangliion cells, the remaining ones being swollen and in various stages of disintegration. The arteries were narrowed and sometimes almost obliterated. (2.) Chronic intoxication in which the animal lived more than ten days. The pupils reacted up to the sixth week. No ophthalmoscopic changes other than pallor of the papilla and narrowing of the arteries. Microscopically the atrophy was more advanced than in the acute cases. The ganglion cells were as in the acute cases, with a solution of the nuclear pigment. The chromatin was disintegrated in many of the cells of the nuclear layers of the retina. He gives a résumé of 8 cases in which there was retro-bulbar neuritis, atrophy, contracted arteries, concentrically contracted fields and blindness. His conclusions relative to the toxic action of the drug upon the eye are: that the amblyopia is due to optic nerve atrophy with primary involvement of the retina; though in certain cases central lesions are shown to be present experimentally and suggested clinically.

This case is, I believe, the only one reported from the United States. It is the second one reported in which the amblyopia seems to have resulted from the use of soamin. The subject is also worthy of our consideration in view of the approaching distribution of Ehrlich's "606". If I am correctly informed, this, too, is a synthetic preparation of arsenic. The world-wide publicity already given to the achievements of this remedy will result in its more or less indiscriminate use by every physician who can get it. If it is as potent as reported, it must possess elements which powerfully influence the human organism. It would be strange indeed if this influence is altogether for good. In accepting and using this remedy, we must not forget that all of its predecessors were, for a time, considered harmless in their action upon the visual apparatus, by authorities in whom we had every right to repose the greatest confidence.

A CASE OF THROMBOSIS OF THE UPPER VENA NASALIS DUE TO SYPHILIS.*

By Mrs. Dr. H. Puscariu,
JASSY, RUMANIA.

Translated by Adolf Alt, M.D.

Michel (*Graefe's Arch.*, 1878) was the first to sever from the many forms of retinal apoplexy the group due to an obstruction of the vena centralis or one of its branches.

Zehender and Angelucci (*Kl. Mtsbl.*, 1878-1880), Weinbaum (*Graefe's Arch.*, 1892), Tuerk (*Thesis, Zuerich,* 1890) confirmed the diagnosis of thrombosis of the central vein by anatomical and microscopical examinations.

Other authors, like Wagenmann (*Arch. f. Ophth.*, 1892) found microscopically an embolism in the central arteries in cases with the ophthalmoscopic signs of thrombosis of the central vein.

Others, like Weinbaum (loc. cit.), Purtscher (*Arch. f. Augenhlk,* 1896), Harms (*X. Congr. f. Ophth.*, 1904) found a thrombosis of vena centralis in other diseases, for instance in hæmorrhagic glaucoma.

Elschnig (*Arch. f. Augenhlk,* 1891) and Yamaguchi (*Klin. Mtsbl.*, 1903) found a thrombosis of the vena centralis in cases of choked disc.

Michel (*Zeitschr. f. Augenhlk,* 1899) found thrombosis in a case in which the ophthalmoscopic signs were like those of a retinitis albuminurica.

From this we must assume that it was impossible to certainly define a nosological type of thrombosis of the vena centralis. In consequence this chapter is wanting in some text-books like the ones of von Wecker, and Galezowski.

Panas, after a short description, states that the thrombosis of the vena centralis cannot be considered as giving a definite and well defined picture. Fuchs as well as Greef in Axenfeld's textbook write but little on this subject.

The cases which have thus far been described concerned patients at an advanced age, from 50 to 80 years. H. Claiborne (*Ophth. Rec.*, 1897) observed it in a woman, 31 years old.

The thrombosis occurs in individuals with arterio-sclerosis, marasmus, senile gangrene, or erisypelas of the face, which first causes an orbital thrombo-phlebitis and then produces thrombosis of the vena centralis.

---

*Klinische Mtsbl., July, 1910, p. 45.

Our case is that of a young man, 28 years old, in whom none of these affections was present, but he had acquired lues three years previously. We could, of course, not compare the clinical symptoms with histological conditions. Yet, the course and the symptoms were so characteristic, that the diagnosis of a thrombosis of the upper vena nasalis retinæ was clear.

Patient, G. V., 28 years old, unmarried, a shoemaker, came on January 29th, 1909, to the ophthalmological clinic at Jassy and complained of a gradual loss of visual acuity in the right eye which had suddenly begun seven days previously.

His father had died 18 years ago from an unknown disease. His mother suffers with an affection of the heart and two brothers of his had died, also, from unknown diseases. From childhood on our patient has suffered from malaria. Three years ago while serving in the army he acquired a chancre which was followed by secondary symptoms.

The treatment had consisted in only ten intramuscular injections of mercury. Five of these were given three years ago and five only one month before the eye became affected. With the loss of vision severe headache had appeared in the right side of the head.

Status præsens.—Patient is well developed and strong. All of the chest and abdominal organs are well. Neither albumen nor sugar in the urine. The pupils are equal. The pupillary reflex is normal in the left eye, somewhat weaker in the right.

V.R.E.=1; L.E. 6/200.

Ophthalmoscopical examination.—In the indirect as well as in the direct image of the right eye the media were perfectly clear with the exception of some opacities in the vitreous body. The optic papilla was dim with veiled margins, especially on the nasal side. The arteries are very small, the veins enlarged. Upwards and inwards two larger and some smaller hæmorrhagic spots are found near the papilla. The veins leading to these are enormously enlarged, dark in color and disappear in the hæmorrhagic spots. Where they emerge they are very pale and hardly recognizable as far as they reach on the papilla. The part of the retina between the hæmorrhagic spots and the papilla is pale, yellowish gray and dim. There is no hæmorrhage in the region of the macula or in any other part of the retina.

In spite of our entreaties the patient would not remain in the hospital, but promised to return after arranging his business. He did, however, not return and we could hear no more of him.

ABSTRACTS FROM MEDICAL LITERATURE

By J. F. Shoemaker, M.D.,

St. Louis, Mo.

THE NOGUCHI SERUM REACTION FOR SYPHILIS AS AN AID TO DIAGNOSIS IN EYE LESIONS.

Albert E. Bulson (*Jr. A. M. A.*, July 16, 1910), after pointing out the difference between the Wassermann test for syphilis and Noguchi's modification, quotes briefly from Noguchi's work a description of the technic of this test. Williams has investigated the value of the serum test for the diagnosis of syphilis and finds a positive reaction in 75 per cent. of over 8,000 tests made by numerous investigators by different methods. The following tabulation of the results is given which demonstrates the value of the serum reaction:

POSITIVE REACTIONS IN 8,000 CASES OF HEREDITARY AND ACQUIRED SYPHILIS.
(Williams)

Stage of Disease.	% Positive.
Primary syphilis	70
Secondary syphilis	89
Tertiary syphilis	78
Early latent (late secondary symptoms)	51
Late latent (following tertiary symptoms)	47
Hereditary syphilis	95
Cerebrospinal syphilis	48
General paralysis	88
Tabes	63

Noguchi claims that his method gives uniformly reliable results, if properly carried out, and it is an improvement over Wassermann's or any other method in that it is not so cumbersome or complicated. Reports seem to show that it is more reliable, also. As, up to the present time, the test does not appear to have been made in very many cases of eye disease, Bulson decided some time ago to take up the work and make the test in cases of eye diseases, especially those of doubtful ætiology, in order to determine the value of the serum test for syphilis in this class of cases. He admits that his findings may not be absolutely accurate because of the lack of long experience in making

the test, but thinks they are of some value and hopes later to report on a much larger number of cases and review the results secured by others. He tabulates a condensed history of twenty-six cases and the results of the serum test. In thirteen of the cases the test was positive and of these thirteen cases ten gave no definite history of syphilitic infection. Seven of them had received antisyphilitic treatment which probably accounts for the weak positive reactions secured in some of them. Of the thirteen negative reactions four were in patients giving a history of syphilitic infection and several others presented decided evidences of secondary manifestations. The active antisyphilitic treatment in these cases undoubtedly accounts for the negative reaction to the serum test. This with the further fact that treatment in a number of the author's cases, which had at first shown a positive reaction, tended to cause a disappearance of the reaction agrees with the assertions of various writers that a short course of treatment often causes the reaction to disappear. The author says:

"In conclusion it may be said that the reaction is destined to be of value to the ophthalmologist as an aid in making a diagnosis, particularly in cases of doubtful origin, and also as a guide to selection of therapeutic measures. But to be reliable and hence of most value the test should be applied to those cases of which a reasonable complete history has been obtained; the reagents employed should be prepared in an accurate and careful manner; the technic as advocated by Noguchi and others should be accurately followed; and the interpretation of the results considered in the light of the experience and skill with which the tests have been made. The reactions should be verified, and control tests should be introduced as often as possible. Furthermore, all things being equal, the results will be most reliable and hence most valuable when done by those with facilities and training for hemolytic work."

THE DANGER OF SYMPATHETIC OPHTHALMIA FROM THE USE OF THE CAUTERY IN TREATING IRIS-PROLAPSE.

OTHER METHODS OF TREATMENT.

H. Gifford (*Jr. A. M. A.*, July 30, 1910), having had sympathetic ophthalmia follow the use of the cautery in treating prolapse of the iris some years ago, has noticed reports of a number of cases of the same kind by such men as Alexander, Darier,

Fuchs and others since, so that he believes there is a real danger in this method of treating iris-prolapse. He thinks that the charred tissue left after the use of the cautery is favorable for the growth and development of germs and that when the cautery is used it is best to scrape away the dead tissue and either cover the surface with conjunctiva or touch the raw surface with trichloracetic acid. He believes the use of this latter agent is preferable in the treatment of prolapse of the iris rather than the cautery.

His conclusions are as follows:

1. Fresh non-infected prolapse should be replaced, if possible; preferably by Dunn's method.

2. Prolapses which cannot be cleanly excised should, if possible, be cauterized and the area scraped and protected at once by a conjunctival flap.

3. On account of the danger of sympathetic ophthalmia no prolapse should be treated by a hot metal cautery, unless a protecting conjunctival flap can be made to adhere to the area cauterized; it is probably safer to let the prolapse alone.

4. In some cases of large corneal prolapses to which conjunctival flaps can be made to adhere with difficulty or not at all, the use of trichloracetic acid (and probably of various other chemicals) produces a firm non-iritable scar. Whether this method is entirely devoid of danger remains to be seen.

TESTS OF VISUAL ACUITY AND CARDS FOR SUBJECTIVE CORRECTION OF AMETROPIA.

Edward Jackson (*Jr. A. M. A.*, Aug. 6, 1910) says:

"The testing of visual acuity and the measurement of ametropia by the improved vision obtained by test-lenses are two distinct processes, which will be best carried out by different forms of apparatus. In testing visual acuity we need only to determine a single 'minimum separabile,' which is done most simply by finding the greatest distance at which a test of known visibility can be recognized. In the subjective measurement of ametropia we have to provide a 'minimum separabile' progressively diminishing as the accurate correction of the refractive error is more nearly approximated. This requires a series of test-objects progressively diminishing in size, such as the ordinary test-cards furnish."

He suggests that this is an appropriate time to consider this subject inasmuch as the Ophthalmologic Congress at Naples last year adopted as the internal standard of normal visual acuity

the ability to recognize two points separated by an angle of one minute; and selected the so-called "broken ring" as the test of this ability. Jackson thinks the broken ring is an improvement over the black square with a space cut out of one side which he suggested in 1890, as the ring can be turned in eight different positions, whereas the square could be turned in only four. A single broken ring on a card can be turned an indefinite number of times while the card is concealed and thus determine accurately the visual acuity. Moreover, the examiner can approach or go farther from the person being tested and thus make a card with a single broken ring on it sufficient to test the visual acuity whether it be good or poor, This makes it valuable where it is desired to make tests away from the office and especially in testing school children. The ordinary test-card is not so accurate a test of visual acuity because some letters are more easily recognized than others of the same size. In the subjective measurement of ametropia letters and numerals are the best test objects as they are easily designated. And here the unequal visibility of letters may be an advantage as a change of lenses will bring out certain letters in a line that could not be recognized at first. Where letters are used for testing visual acuity, their visibility should be compared with that of the international standard broken ring, as otherwise we cannot be accurate.

CONCOMITANT SQUINT WITH SOME REMARKS ON ITS ÆTIOLOGY AND TREATMENT.

A. E. Davis (*Jr. A M. A.*, Aug. 16, 1910) discusses the different theories of the cause of concomitant squint, viz.: the muscle theory, the accommodation theory and the fusion theory. The muscle theory, that is that the defect is a local one entirely and due to a certain muscle being too short, was the first plausible theory advanced which led to any rational or effective treatment. This theory led to the practice of cutting the muscle that was supposed to be at fault. It has been almost entirely abandoned. The accommodation theory was elaborated and advanced by Donders in 1864, and was generally held to be the correct one by the medical profession for over a quarter of a century. In 1893 Worth undertook to show that hypermetropia was but a contributing cause and that the essential cause of convergent squint is a faulty development of the fusion faculty.

Davis believes that neither the accommodation theory nor the fusion theory will alone explain all cases and in fact the two

theories combined fail to solve some of the questions in certain cases. There is much truth in both, however, and taken together they account for most cases. In divergent squint the fusion theory fails utterly as this form of squint usually appears after the age of seven years when the fusion faculty is supposed to be fully developed. The accommodation theory is more applicable, although Donders called attention to certain other factors which are now generally recognized as entering into the cause of divergent squint. These are, first, an original preponderance of the external recti muscles; second, smallness of the angle alpha; third, the favorable form of the eye for outward movement. The author thinks it is well to look at the subject from a double viewpoint—the physical and the psychical—in trying to recognize and harmonize the "accommodation" and "fusion" theories. He says:

"The accommodation theory, while it gives to the fusion sense a secondary position, emphasizes the physical side of the squint; for example, (1) errors of refraction, especially in cases in which that error is greater in one eye than in the other; (2) opacities in the refractive media or lesions of the fundus enfeebling the sight in one eye; (3) the variation in the angle of visual lines to the corneal optic axis of the eye—the angle alpha of Donders, or gamma of Landolt and later writers; (4) paresis of accommodation; (5) length of the eyeball; (6) anatomic defects in the structure or insertion of the straight muscles, etc. The fusion theory emphasizes the psychic phase of squint; for example, (1) faulty development of the fusion sense, or the power to fuse the images of the two eyes into one, with the resultant loss of binocular vision; (2) the psychic phenomenon of suppression of the image or one eye, especially liable to occur when the vision in said eye is reduced from any cause, (this, however, may occur when the vision is perfect in each eye, when the fusion sense is supposed to be congenitally absent.)"

The treatment of squint is discussed under two headings; first, non-operative or orthoptic treatment; second, surgical treatment. Orthoptic treatment consists of (1) correction of the error of refraction; (2) use of the occlusion pad; (3) the use of cycloplegics in the fixing eye; (4) stereoscopic exercises for training the fusion sense. The author discusses these separately. He thinks, however, that one-half to two-thirds of the cases of convergent squint are not cured by orthoptic treatment. Then the question of surgical treatment comes up, and Davis believes it is wise to operate as soon as the angle of squint ceases to improve under orthoptic treatment. The advantages of "advancement"

and "tenotomy" are discussed as also the different methods of doing these operations, and the author states his preferences, which are the straight advancement operation and Pana's tenotomy operation. Whether advancement or tenotomy should be done he thinks depends largely upon the case.

THE SURGICAL TREATMENT OF SEPARATION OF THE RETINA.

Casey A. Wood (*Jr. A. M. A.*, July 23, 1910) says:

"One who has made a study of the observations of others in the surgical treatment of detached retina and has added to it his own experience—though that may be limited—will, I think, come to these conclusions:

1. Inasmuch as separation of the retina is not a distinct disease but merely one sign—albeit a very important one—of several different affections, it is not to be expected that it is to be cured or much relieved in very instance by some particular operation. The 'one-disease-one-operation' idea can have no place here.

2. A large percentage of retinal repositions, including an unknown proportion of those that follow operative measures, belong to the class of spontaneous cures. In such cases relief may have been assisted, hastened or rendered more or less permanent by the remedies exhibited, but the probability is that the patients would have recovered in any event.

3. So far as prognosis is concerned, the more hopeful cases are the recent, limited varieties—those produced by traumatism, postretinal hæmorrhage and the like—as well as those resulting from removable causes. *Per contra,* old, extensive detachments, especially when associated with marked degeneration of the retina, vitreous and chorioid, are not likely to get well under any forms of treatment. A long-separated, starved retina rarely regains its lost functions.

4. Recurrence of the detachment forms a disappointing feature in the treatment of the disease, and this fact should be considered by both patient and surgeon when the subject of operation is broached. On the other hand, it has been abundantly demonstrated that patients with separated retina have recovered after several relapses and after having submitted to many operations.

5. No patient should be regarded as permanently cured until at least a year after the replacement of the detached membrane. It is true that relapses are recorded after an interval of several years, but they are unusual.

6. When a patient presents himself it is best to try for, say a month—indefinitely as long as improvement continues—non-operative measures. A thorough study should be made of the case to determine, if possible, the cause of the detachment, that it may be treated *secundum artem* and, perhaps, removed. With this causal treatment give subconjunctival injections, instil atropin and keep the patient in bed. Pilocarpin sweats, with iodids or sodium silicylate, are also generally indicated.

7. Failing to improve vision or to replace the separated retina by milder means, resort should be had to operation, and the question of the best operation for the case in hand at once arises. We know that the function of the retina gradually weakens the longer it is displaced; consequently the sooner one makes a choice of operation the better.

8. Deutschmann advises against his operation as long as the postretinal fluid is held within the upper quadrants of the globe. If we are debarred by this circumstance from the use of his methods, there can be no objection to the employment of scleral puncture, combined with punctiform cautery of the denuded sclera over the site of the detachment. Why should we wait until the retina is further detached and degenerated?

9. In those cases in which the sac occupies, as it generally does, the lower aspect of the hyaloid chamber, Deutschmann's method of bisection should be the operation of election, whether or not there be evident rents in the retina or visible fibrillæ in the vitreous.

10. Two weeks after an intrabulbar operation a careful examination of the eye should be made—with the electric ophthalmoscope (so that the patient may keep the prone position), hand perimeter, ward charts, etc.—to decide if improvement has taken place in the local conditions as well as in central and peripheral vision.

11. Assuming the eye to have recovered from any operative measure, i. e., to be free from either intraocular or extraocular inflammation, the same or another operation may be done in from three to six weeks' time.

12. In unpromising cases Deutschmann's intraocular injections of animal vitreous is in order, although Mueller's exsection of the sclera seems a rational though formidable procedure, which an improved technic and a wider experience may yet demonstrate to be of great value in the conduct of this extremely serious condition."

BOOK REVIEWS.

Die mikroskopischen Untersuchungsmethoden des Auges. (The methods of microscopical examination of the eye.) By Dr. S. Seligmann. 2nd edition. Berlin, 1911. S. Karger.

When the first edition of this very useful book had come into our hands in 1899 we hastened to recommend it most heartily to our readers. It is with equally great pleasure that we welcome this second edition, which includes everything which has been added to our knowledge since then. A worker in the field of histology and histopathology of the eye should not be without this book, which will prove of great help to him.

Traitemant du Glaucom Chronique (Sclérecto-iridectomie et sclérectomie simple). [The treatment of chronic glaucoma. (Sclerecto-iridectomy and simple sclerectomy).]. By Dr. J. Beauvieux. Paris, 1910. Steinheil.

The author, a pupil of Lagrange, desires to show the superiority of the Lagrange operation for chronic glaucoma over the other methods recommended and employed before. After a chapter on the pathology of chronic glaucoma, he discusses first the non-operative and then the operative methods of treatment of this disease. That Lagrange's end to produce a lasting opening in the sclera covered by conjunctiva only is realized is shown in a number of good illustrations. He gives a detailed description of 44 cases so treated and in the conclusions attests the superiority of the Lagrange operation over any previous one in a very convincing manner.

The Refractive and Motor Mechanism of the Eye. By W. N. Souter, M.D. With 148 illustrations. The Keystone Publishing Co., Philadelphia, 1910. Price $2.00.

A thoroughly good book on a subject which, on account of its importance, bears repetition well. Although in perusing it we find nothing strikingly original, the book can with safety be recommended. Alt.